rectal examinations while showering. In the author's practice, this suggestion to homosexual patients has proved to be a highly effective means of accurate discovery of the existence and location of condylomata, or of a recurrence following original treatment.

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The Use of the Oxytocin Challenge Test in High-Risk Pregnancies

THE OXYTOCIN CHALLENGE TEST (OCT; also called the contraction stress test) is an attempt to duplicate the stresses of labor by inducing contractions and observing the fetal heart response. This test has been widely accepted in the United States as a useful clinical method of assessing fetal well-being in the hazardous intrauterine environment encountered in certain high-risk pregnancies.

The test consists of intravenous administration of oxytocin by an infusion pump sufficient to produce three contractions during a ten-minute interval, and the simultaneous recording of fetal heart rate and uterine contractions using an electronic fetal monitor. When clinically indicated, the test can be carried out as early as in the 32nd week. The test can be done without oxytocin when contractions are occurring spontaneously.

This test is contraindicated in cases of suspected labor before term, vaginal bleeding and cervical incompetence. Relative contraindications include previous cesarean sections or multiple gestation when a premature fetus would compound the survival risk factor.

A positive test result is determined by a fetal monitor recording of late or variable decelerations of fetal heart rate at, or just beyond, a uterine contraction. The pattern should be repeated with most subsequent uterine contractions or with each of three contractions with an interpretable heart rate during a ten-minute period. Positive findings, if not acted on, are associated with a high incidence of fetal death or fetal distress.

A negative test finding is clearly the greatest benefit of the OCT and is observed in about 90 percent of cases. A negative response is interpreted when uterine contractions occur at a frequency of three in ten minutes with no decelerations of fetal heart rate. To maintain surveillance on the uterofetal-placenta unit, a negative test may be repeated at intervals of a week or less.

A suspicious result is shown by definite, but inconsistent, late decelerations failing to persist with most uterine contractions. After a suspicious test result, it is advisable to repeat the test in 24 hours.

The test is considered unsatisfactory if the quality of the recording is technically poor enough to prevent determining whether decelerations are present, or if there are fewer than three contractions in ten minutes.

The test requires 90 to 120 minutes and should be done with meticulous care by trained nursing and medical personnel.

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Parathyroid Hormone and Calcium Abnormalities

THE GREATER AVAILABILITY of laboratory analyses of chemical fractions of the blood of patients seen by family physicians has made it possible to diagnose hypercalcemia in patients who may not have presented with the clinical features of primary hyperparathyroidism. The parathyroid hormone (PTH) assay is also available and may be added to the battery of tests used in the evaluation of clinical problems.

The specificity of the parathyroid hormone analysis is sufficient to recognize fragments and intact PTH. However, in some 15 percent to 20 percent of patients there is sufficient overlap, in which it is possible to have high PTH production without primary hypothyroid adenoma, parathyroid hyperplasia or parathyroid carcinoma. The source of the ectopic production of the parathyroid hormone in these cases is usually a malignant lesion. The changes noted in hyperparathyroidism, notably hypercalcemia, hypophosphatemia and elevated levels of alkaline phosphatase, as well as the clinical symptoms of debilitation or constitutional signs of a malignant condition, may be extremely important in differentiating the

source of elevated PTH. Generally, patients with malignant lesions tend to have higher levels of serum calcium than patients with primary hyperparathyroidism; the PTH levels are lower in patients with malignant lesions in contrast to levels noted in patients with primary hyperparathyroidism; and high levels of calcium, are attained more rapidly than in the chronic and slowly progressive disorder that marks primary hyperparathyroidism.

In the differential diagnosis of hyperparathyroidism, other sources of hypercalcemia need to be considered. These may include, in addition to neoplasms with or without metastasis, multiple myeloma, sarcoidosis, thyrotoxicosis, immobilization in the young, Paget disease, vitamin D intoxication, milk-alkali syndrome, Addison disease and the use of thiazide diuretics.

A workup by physicians in family practice for a patient who has hypercalcemia should include a careful history; complete blood count; studies of sedimentation rate, electrolytes (including serum), magnesium levels, protein electrophoresis and thyroid function; x-ray study of the chest; an intravenous pyelogram, and skeletal survey x-ray studies. When the association among hypercalcemia and hypophosphatemia and increased alkaline phosphatase is noted, and confirmed on repeated examinations, the immunoassay test for parathyroid hormone may be indicated. Additional tests which may be useful include measurement of tubular absorption of phosphate after either calcium or phosphate infusion.

If serum calcium levels are greater than 15 mg per dl hypercalcemia is severe. When the patient is symptomatic, a short QT interval may be noted on the electrocardiogram, and this requires restricted calcium intake, hydration, forced diuresis with furosemide and ethacrynic acid. In long-term management, therapy with steroids may be indicated as well as oral administration of phosphates. Dialysis may be considered in a life-threatening situation in which the use of other measures is restricted. Mithramycin inhibits bone resorption and lowers the calcium level when given intravenously in a dose of 25 mg per kg of body weight.

Patients who have increased calcium levels and no evidence of clinical disease should be observed closely for the development of clinical or chemical abnormalities that may indicate progression of hyperparathyroidism.

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